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CORRESPONDENCE

The endpoint of fluid responsiveness: is cardiac output enough?

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We read with great interest the editorial by Professors Monnet, De Backer, and Pinsky reviewing the usage of ventilator to predict fluid responsiveness in shock [1]. They reviewed fluid responsiveness evaluation strategy by ventilator-induced cardiopulmonary interactions, however, as the golden parameter to predict fluid responsiveness—cardiac output is enough, or not?

As the mainstay therapy of shock, fluids is given to increase cardiac output in order to improve key organ and tissue perfusion, and the latter might be more important. However, the classical parameters of fluid responsiveness are the change of cardiac output after the fluid bolus, passive leg raising or ventilator-induced cardiopulmonary interactions, even though better perfusion responsiveness to fluids matters a lot.

Cardiac output variations are not always associated with peripheral or organ tissue perfusion. In our study performed in shock patients, even with increased cardiac output, the transcutaneous partial pressure of oxygen ($PtcO_2$), which detect tissue ischemia or inadequate perfusion earlier, might be low, and two patients who had a $PtcO_2$ less than 40 mmHg at baseline with a relatively normal or high cardiac output did not survive [2].

Microcirculation is responsible for regulating tissue perfusion to meet oxygen requirements. Tissue oxygenation depends on adequate arterial oxygenation and total blood flow, diffusion of oxygen across capillaries and the distribution of microcirculatory flow. In patients with microcirculatory dysfunction, even cardiac output increased, tissue perfusion might not be changed. It is reported [3] that in delayed resuscitation situations, even fluid resuscitation enhancing cardiac output, microcirculation and tissue perfusion are not improved. Another study reported that in septic shock patients with previous hypertension, increasing the dose of norepinephrine bring in cardiac output and microvascular improvement, but the relationship between the changes of cardiac out and microvascular variables are not related [4].

Meanwhile, organ dysfunction is an integral part of shock, but the pathophysiology involves complex hemodynamic mechanisms, the alterations of perfusion in each organ is different, e.g., the perfusion of brain and gastrointestinal tract are totally different in early and late phase of shock. More attention is needed when organ or peripheral tissue perfusion not link to cardiac output, and organ- or tissue-specific perfusion improvement might be the future research priorities.

We propose that it may be time to move beyond the simplified concept of "fluid responsiveness" by the increase of cardiac output to "perfusion responsiveness", with the new technology era [5].

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Data availability
All data generated or analyzed during this study are included in this published article.

Conflict of interest
The authors declare that they have no competing interests.

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